
A new anabolic compound, LLP2A-Ale, reserves periodontal bone loss in mice through augmentation of bone formation.

Journal: BMC Pharmacol Toxicol

Publication Year: 2020

Authors: Min Jiang, Lixian Liu, Ruiwu Liu, Kit S Lam, Nancy E Lane, Wei Yao

PubMed link: 33187558

Funding Grants: Treatment of non-traumatic osteonecrosis with endogenous Mesenchymal stem cells

Public Summary:

BACKGROUND: Currently, there are no effective medications to reverse periodontal disease (PD)-induced bone loss. The objective of this study was to test a new anabolic compound, LLP2A-Ale, or with the combination treatment of mesenchymal stromal cell (MSC), in the treatment of bone loss secondary to PD. **METHODS:** PD was induced in mice by placing a ligature around the second right molar. At one week after disease induction, the mice were treated with placebo, LLP2A-Ale, MSCs, or combination of LLP2A-Ale + MSCs, and euthanized at week 4. **RESULTS:** We found that PD induced alveolar bone loss that was associated with reduced bone formation. LLP2A-Ale alone or in combination with MSCs sustained alveolar bone formation and reversed alveolar bone loss. Additionally, PD alone caused systemic inflammation and increased the circulating levels of G-CSF, IP-10, MIP-1a, and MIP2, which were suppressed by LLP2A-Ale +/- MSCs. LLP2A-Ale +/- MSCs increased bone formation at the peripheral skeletal site (distal femur), which was otherwise suppressed by PD. **CONCLUSION:** Our findings indicated that LLP2A-Ale treatment rescued alveolar bone loss caused by PD, primarily by increasing bone formation. LLP2A-Ale also attenuated the circulating levels of a series of inflammatory cytokines and reversed the PD-induced suppression of systemic bone formation.

Scientific Abstract:

BACKGROUND: Currently, there are no effective medications to reverse periodontal disease (PD)-induced bone loss. The objective of this study was to test a new anabolic compound, LLP2A-Ale, or with the combination treatment of mesenchymal stromal cell (MSC), in the treatment of bone loss secondary to PD. **METHODS:** PD was induced in mice by placing a ligature around the second right molar. At one week after disease induction, the mice were treated with placebo, LLP2A-Ale, MSCs, or combination of LLP2A-Ale + MSCs, and euthanized at week 4. **RESULTS:** We found that PD induced alveolar bone loss that was associated with reduced bone formation. LLP2A-Ale alone or in combination with MSCs sustained alveolar bone formation and reversed alveolar bone loss. Additionally, PD alone caused systemic inflammation and increased the circulating levels of G-CSF, IP-10, MIP-1a, and MIP2, which were suppressed by LLP2A-Ale +/- MSCs. LLP2A-Ale +/- MSCs increased bone formation at the peripheral skeletal site (distal femur), which was otherwise suppressed by PD. **CONCLUSION:** Our findings indicated that LLP2A-Ale treatment rescued alveolar bone loss caused by PD, primarily by increasing bone formation. LLP2A-Ale also attenuated the circulating levels of a series of inflammatory cytokines and reversed the PD-induced suppression of systemic bone formation.

Source URL: <https://www.cirm.ca.gov/about-cirm/publications/new-anabolic-compound-llp2a-ale-reserves-periodontal-bone-loss-mice-through>